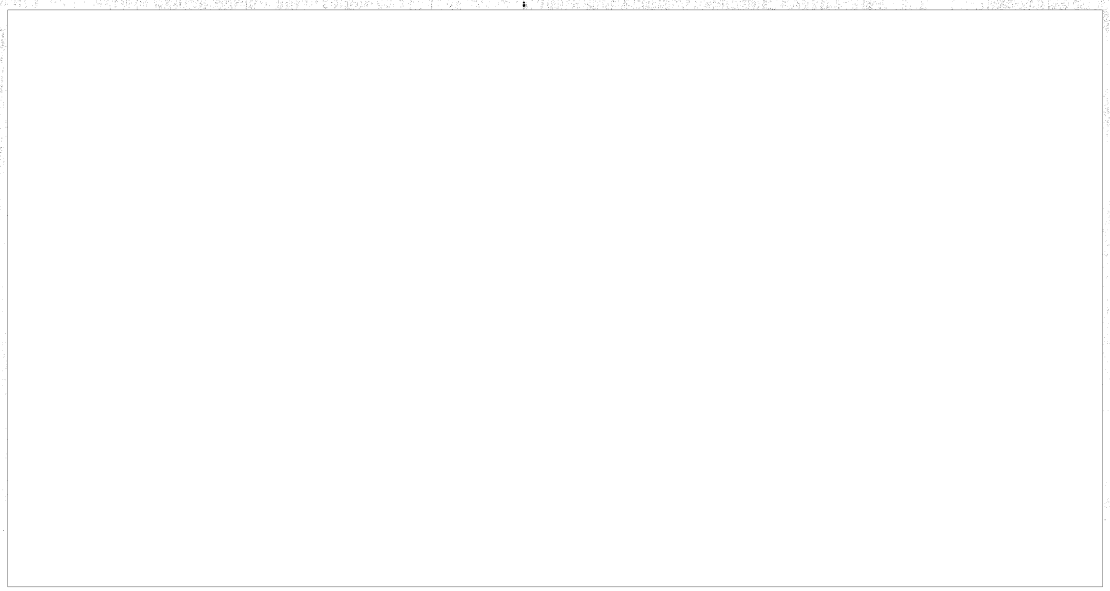
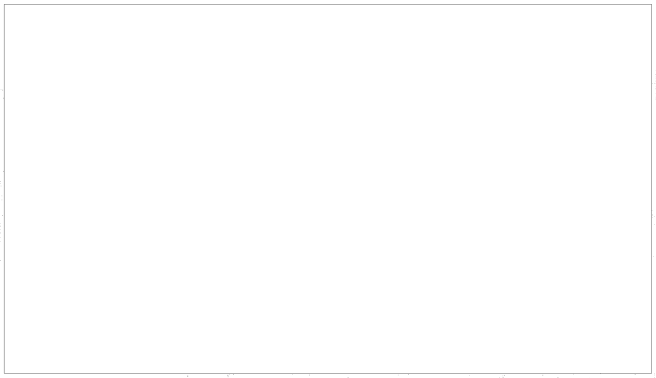


Biologic Properties of Tenchous Disease

(Nadyezna Waterynarzina, Antonin Klobouk,

Warsaw, October 1950)



BIOLOGIC PROPERTIES OF TESCHEN'S DISEASE

Antonin Klobouk

From the Pujatric Veterinary School in Brno,
Director Professor Doctor Antonin Klobouk

The contagious Teschen's disease is presently the most common disease of swine in the Republic of Czechoslovakia.

According to official statistics, as of 15 May 1950 there were 243 districts (2,966 villages and 12,357 farms) in which the epidemic was brought under control.

In 1926 I suggested to the Ministry of Agriculture, under whose jurisdiction the entire Veterinary Service Organization came at that time, that veterinary regulations be issued to counteract the spread of this disease, as its contagious nature was already well established and its etiology known (virus).

Up to 1936, there was no knowledge of the means by which the disease was transmitted because the diagnosis was frequently mistaken. When I had first come into contact with this disease in the years 1913-1914 I was convinced that this was a specific ailment and considered it a toxic alimentary condition of unknown origin.

It is clear that the specificity of this disease has become more obvious in the course of meticulous research work investigating the correlation between the clinical symptoms and anatomical changes observed, particularly when a histological examination of the central nervous system was introduced as a means of corroborating the clinical diagnosis.

A systematic study of this disease began in 1930, many doubtful questions have been cleared up; many more problems, however, remain unsolved.

From the very beginning it was noticed that the contagiousness of the disease was relatively low. Its spread can be prevented by relatively simple hygienic measures. Despite this, it has been observed that in some regions almost an entire livestock population can be affected. It happens mostly on small farms when ~~conditions are unfavorable (poor~~ hygienic conditions ~~)~~.

From the epidemiological point of view, it is important to accumulate all available data pertaining to the circumstances under which the disease is spreading.

Practical observation and much experimentation allow us to conclude simply that the sick animal is the primary source in spreading the infection; so also are the slaughtered hog's inner organs which during the process of slaughter might have come into contact with the virulent cerebrospinal fluid or tissue.

Furthermore, bearing in mind similar experimental findings with other diseases, we cannot discard the hypothesis that some seemingly healthy animals might be carriers. At present, it is difficult to say how high the percentage of such carriers might be. Up to this date, no convincing experimental evidence has been found to support the hypothesis that this means of spreading the disease is a real possibility.

Personally, I am inclined to believe that the percentage of such animal carriers is small. This opinion has been corroborated by the fact that many breeding centers report no

reoccurence after the epidemic has subsided; moreover, the disease has not spread to other production centers to which carefully selected and thoroughly examined specimens from the formerly affected farms have been sold for breeding purposes.

The possibility of communicating this disease by indirect contact has not been sufficiently explored. It is difficult to infect an animal with this disease experimentally, even by close contact. That is why simple hygienic measures of precaution are effective in the prevention of this disease. However, the fact that Teschens disease has been recently spreading and affecting larger areas seems to contradict this statement.

It would be desirable to follow closely the paths of invasion chosen by the infection, and for this purpose we must concentrate our attention on what finally happens to the virus within as well as outside the animal's body. The importance of this problem has been emphasized by me from the very start of research investigation on Teschens disease.

RESEARCH ON DETECTION OF VIRUS IN BLOOD

I have stressed the importance of clarifying the problem of the presence of Teschen's disease virus in blood and of determining the amounts, it occurs in the blood as compared with the central nervous system.

Clarification of this matter is of great importance for tracing the infection route of the virus in the sick organism, and is also essential for determining whether the virus of Teschen's disease actually possesses neurotropic properties, or whether this idea should be rejected.

reoccurence after the epidemic has subsided; moreover, the disease has not spread to other production centers to which carefully selected and thoroughly examined specimens from the formerly affected farms have been sold for breeding purposes.

The possibility of communicating this disease by indirect contact has not been sufficiently explored. It is difficult to infect an animal with this disease experimentally, even by close contact. That is why simple hygienic measures of precaution are effective in the prevention of this disease. However, the fact that Teschens disease has been recently spreading and affecting larger areas seems to contradict this statement.

It would be desirable to follow closely the paths of invasion chosen by the infection, and for this purpose we must concentrate our attention on what finally happens to the virus within as well as outside the animal's body. The importance of this problem has been emphasized by me from the very start of research investigation on Teschens disease.

RESEARCH ON DETECTION OF VIRUS IN BLOOD

I have stressed the importance of clarifying the problem of the presence of Teschens disease virus in blood and of determining the amounts it occurs in the blood as compared with the central nervous system.

Clarification of this matter is of great importance for tracing the infection route of the virus in the sick organism, and is also essential for determining whether the virus of Teschen's disease actually possesses neurotropic properties, or whether this idea should be rejected.

As far back as in 1934, with this aim in view, I did some experimental work: twice I withdrew blood from animals affected with typical Teschen's disease at the prodromal stage; the blood was defibrinated and injected intracerebrally to two young pigs in amounts of 0.5 cubic centimeters and 1 cubic centimeter; it was administered subcutaneously ~~as well~~ also in amounts of 40 and 60 cubic centimeters. The experiment was performed on 4 young pigs altogether. Their blood was taken when they were feverish, their respective temperatures being 41.2, 40.6, 41.2, and 41 degrees centigrade. The blood specimens were taken from 3 to 18 hours after a temperature of 40 degrees or more was noted. The blood specimens were defibrinated or mixed with a 10 percent solution of sodium citrate in proportion 1:5. The blood was injected to the experimental pigs within one to eight hours after it had been taken. Thus, the amount of blood injected into the brain or subcutaneously was considerably large, and yet none of the animals took sick or showed any sign of Teschen's disease.

Similarly, many other investigators who studied this problem later on failed to receive positive results by using blood of sick animals experimentally.

Shaefer and Heynen studied the blood of 4 young pigs who were artificially infected with the Teschen virus by different methods. They withdrew the blood of sick animals at 24-hour intervals and examined it for the presence of the virus by transferring it to a healthy suckling pig.

They failed to induce an infection by this method. Only in the case of 1 pig was there an elevation of temperature; it lasted 3 days and the authors were inclined to consider the infection as a mild one of short duration, leaving only slight resistance. At

~~a mild one of short duration, leaving only slight resistance. At~~ the following reinfection attempt this resistance manifested itself by a considerably prolonged incubation period and a spontaneous sterilization of the central nervous system.

Fortner experimented with blood from a considerable number of sick suckling pigs; his attempts to infect 15 pigs failed -- none of the pigs showed any signs or symptoms of Teschen's disease. On reinfection, 9 of the 15 specimens took sick, 6 remained in good health. It is the author's opinion that reinfection by the homogenous method might in some cases produce a so-called silent infection with a resulting resistance.

According to Mussemeier, Gerlach was the only investigator to succeed in finding the Teschen virus in the blood stream of sick animals, even at the initial stage of disease.

A comparison of the amounts of blood with the amount of cerebrospinal fluid emulsion used by me during experimentation procedures will permit ~~the arrival at~~ some very interesting and important conclusions. When cerebrospinal fluid was used for intracerebral inoculation, very small amounts of emulsion, (0.0025 cubic centimeters), produced symptoms of a typical disease the course of which was in some cases grave and even fatal; but amounts of blood four hundred times larger (1 cubic centimeter), introduced ~~by~~ intracerebral ~~route~~ failed to reproduce the disease in 8 experimental cases. A simultaneous subcutaneous administration of blood in amounts of 40 and 66 cubic centimeters had no effect on the experiment. We must therefore emphasize the particular nature of ~~the~~ Teschen's disease as compared with other diseases of swine.

This argument does not permit us, of course, to draw the final conclusion that no virus exists in the blood of sick animals. What we can accept is that the amount of it, if any, in the blood is rather small and at any rate inadequate to produce a successful experimental transmission of the disease by intracerebral administration. Experimental work must, of course, be continued until we have determined the degree of the virus virulence in the blood at every stage of disease.

PRESENCE OF THE VIRUS IN URINE

As far back as ~~the years~~ 1934-1937 I experimented on 19 suckling pigs in an attempt to transmit this disease by urine, but had no positive results. None of the experimental pigs showed signs resembling those of the Teschen disease.

There was one case which has never been clarified -- a suckling pig after having received 2 cubic centimeters of urine (filtered through Berkefeld's filter) intracerebrally and 15 cubic centimeters of same urine specimen subcutaneously died within 12 hours. The urine specimen was collected on the sixth day of the disease.

There was considerable redness at the site of the subcutaneous injection. As the dose of urine administered intracerebrally was large (2 cubic centimeters), I am inclined to believe that the large amount of urine administered in the instance cited produced a severe toxic effect upon the central nervous system.

Fortner, in 1940 and 1941, also did not succeed in reproducing the disease by means of urine. Thus, the same can be said

with reference to urine as was said about blood. Though efforts to transmit the disease experimentally by using urine remained unsuccessful, the possibility of the virus being eliminated through the urinary tract cannot be completely ruled out. At least, one conclusion can be derived from previously negative results -- that the concentration of virus in urine may be so small as to be inadequate for inducing the disease experimentally.

VIRUS VIRULENCE OF INNER ORGANS AND [redacted] FLESH
(except brain and spinal cord)

Obviously, this problem is of tremendous practical importance. The fundamental question is this: do different organs and meat contain the virus, and if so, in what quantities? Since the presence of the virus in blood has never been established, at least not in quantities large enough to induce experimental transmission of the infection, it would be logical to assume that organs and meat would not contain the virus in concentrations suitable for detection (except in the brain and spinal cord, of course).

Feeding with diseased meat, blood, and inner organs of hogs has not produced morbid effects when the brain and spinal cord have been previously carefully removed so as to prevent their making contact with other organs.

This conclusion may have a bearing on the final solution of the problem as to whether we can accept as fact the existence of neurotropic properties of the virus of Teschen's disease. This conforms with the fact that it is so extremely difficult to prove the contagiousness of blood in Teschen's disease. This proof has

been established, however, under special conditions which do not occur in uncontrolled situations, because in practice we are dealing with meat which during the slaughtering process has come into contact with brain and spinal cord tissues.

For this reason, hog meat received from slaughterhouses must be regarded as practically contagious notwithstanding the fact that scientific research carried out by us as well as by other investigators has failed so far to reveal evidence of a contagious agent being present in meat, blood, or inner organs of sick animals.

INFECTIOUSNESS OF THE GASTRO-INTESTINAL TRACT CONTENTS

To the problem of the degree of infectiousness of the contents of the gastro-intestinal tract I personally devoted little time, and I did not arrive at any definite conclusions.

However, Fortner found evidence of the virus being present in feces in 4 cases in which these fecal specimens produced a clinical picture of Teschens disease. This valuable observation proves that next to meat the gastro-intestinal tract of the hog is the most important agent in the transmission of the disease. Investigations made to date seem to indicate that the transmission of this disease by animals takes place through secretory and excremental material. Consequently, the utmost importance of enforcing thorough disinfection of the source of infection becomes self-evident. Wherever sick animals are isolated from pigsties and a thorough disinfection carried out, a rapid extinction of the epidemic may be expected. The truth of this statement has been corroborated by practical experience. Such an action cannot be carried out successfully on small farms where poor hygienic conditions prevail.

INFECTIOUSNESS OF MUSCLE TISSUE, SCIATIC NERVE, BILE, AND SALIVARY GLANDS

Fertner, who studied this problem, could not discover the presence of virus in these tissues nor in their secretion; in particular, he could not achieve a positive result while testing the nasal mucus in 2 cases for the presence of Teschens disease virus during the first 2 days of the paralytic stage of the disease.

INFECTIOUSNESS OF THE VIRUS DERIVED FROM THE CENTRAL NERVOUS SYSTEM WHEN ADMINISTERED ORALLY

Attempts to transmit the infection by oral administration of brain or spinal cord tissue have not given uniform results. In my experiments, 36 percent of the animals fed with scrapings of brain and spinal cords succumbed to the disease. A similar morbidity rate can be observed in large farm units under practical conditions. This proves that a considerable percentage of hogs have a natural resistance.

For experimental work as well as for the purpose of obtaining the most virulent cerebrospinal suspension possible, the following phenomenon is of great practical value: the role of the time factor, with the virulence of brain and spine gradually decreasing during the course of disease, weakening in the later stages, and ultimately disappearing altogether.

According to my experience, a high infectious power of the cerebrospinal substance can be expected within 24 to 48 hours after the appearance of clinical symptoms.

The virulence of this substance drops after 7 days, and from 2 to 3 weeks later the virulence is no longer high enough for the production of vaccine, and too low to allow experimental identification of the virus in the central nervous system.

IMMUNITY AFTER RECOVERY

After the disease has subsided animals become immune against this infection. Experiments show that they cannot be reinfected by the intracerebral route. It is natural that, as with other contagious diseases, attempts have been made to find a working method of preventive immunization against Teschens disease.

EXPERIMENTAL IMMUNIZATION

I have been studying the problem of active immunization against Teschen's disease ever since 1943. The inoculation substance ~~I took~~ was taken from the brain and spinal cord tissues, partly with an addition of 0.5 percent carbolic acid, which gradually reduces the virulence of the virus, and partly with the addition of a physiologic solution only. In the course of my former work I concluded that hogs are resistant to subcutaneous administration of a virulent suspension of cerebrospinal tissue. It is in general very difficult to infect a healthy animal by subcutaneous administration of such a suspension.

My first immunization experiments were performed from 1933 to 1935 on 34 suckling pigs. Of this number 10 were immunized, the remaining 24 serving as a control group.

Method of immunization:

1 subcutaneous injection administered to 3 pigs
2 " " " to 2 pigs
4 " " " to 3 pigs
5 " " " to 2 pigs
6 " " " to 1 pig

Control of degree of immunity was carried out between the 29th and 57th days after inoculation. When a number of injections were given, the time was counted from the day of the first injection. A control reinfection was tried with all inoculated pigs by means of injecting a virulent emulsion of cerebrospinal tissues. Each animal of the experimental as well as the control group received an intracerebral dose of 2.5 milligrams of the substance; this corresponded to 0.5 cubic centimeters of a 1 percent cerebrospinal emulsion.

All the animals of the control (uninoculated) group developed severe symptoms of the disease. The result of these experiments can be summarized as follows: of the 10 inoculated, complete immunity developed in the 5 suckling pigs who received a total of about 3 grams or more of the cerebrospinal substance in several injections. The pig which received the smallest dose (5 milligrams) took severely ill and died on the seventh day after the experimental infection. The pig that received a dose of 50 milligrams also took sick, but not so severely. Of the 3 suckling pigs which were given 100, 100, and 2000 [sic] milligrams of the cerebrospinal substance, two had mild cases of the disease and recovered. The third showed no sign of disease. These experiments led us to the conclusion that only large doses of the cerebrospinal substance administered subcutaneously can produce a state of complete immunity and insure

resistance against intracerebral inoculation of 2.5 milligrams of a virulent cerebrospinal substance. This experimental work done from 14 to 19 years ago became the starting point of further studies on preventive immunization.

At the time that our work was devoted to the problem of etiology of this new disease, preventive inoculation was practiced only in pigsties stricken with Teschen's disease. It can be immediately realized that under such circumstances it was by no means easy to obtain exact data on the effectiveness of this method, as at that time we had no adequate knowledge of the means by which the disease spread, nor was there any definite plan of action against Teschen's disease. During the war, no research work was permitted; and it was not resumed until 1945.

The requirements of practical life forced us to resume work on the problem of preventive immunization. During recent years, new types of vaccine, in particular the absorbtion type, have been produced.

Publications that report the results of applying these vaccines in practice are incomplete, ~~that~~ which is why a final appraisal of their practical value cannot be made at this time. Experimental research on these vaccines, as well as attempts made so far to apply them in practice, are reassuring and justify the hope that the fight against Teschen's disease will give positive results.

With reference to the anti-Teschen's vaccine, I wish to rectify some information published in the professional journal Medycyna Weterynaryjna (1949, page 119, column 1, lines 43-48), specifically, to the effect that I have never prepared an anti-influenza vaccine for

practical use in hog breeding. For the preparation of the anti-Teschen's vaccine I have used the method which worked out as early as 1933.

THE BLOOD SUGAR LEVEL IN TESCHEN'S DISEASE

At the Bujatric clinic of the Veterinary College at Brno, Wywozil conducted studies in 1935 on fluctuations of the blood sugar level in Teschent disease. He observed an increase of the sugar level. This level tended to rise with the progress of the disease. During the incubation period the blood sugar level, as compared with healthy pigs, was on the average 82.48 percent milligrams. In advanced stages of the disease the blood sugar level went up to 103.4 percent milligrams.

The author attributes the increase of the sugar level to stimulation of the sugar center in the medulla oblongata where inflammatory changes are caused by the virus.

RELATION OF TESCHEN'S DISEASE TO OTHER DISEASES

A historical review is necessary to the understanding of this extremely important problem. Teschen's disease, as already mentioned, has been mistaken for other diseases. It was frequently diagnosed as a food poisoning of unknown origin. In retrospect, I am positive now that I observed this disease several times in Przerow during ~~the years~~ 1913 and 1914. At that time I thought it was food poisoning, and even later, 1919 to 1929, I described this disease as food poisoning while teaching at the Veterinary College in Brno.

There have been many other teories concerning the etiology of this disease. If sick animals happened to be simultaneously

affected with ascariasis, the disease was described as a result of a parasitic invasion, and such a diagnosis was not infrequently made by veterinary diagnostic laboratories for lack of other objective findings. These, however, could have been easily discovered if an autopsy had been performed.

When symptoms resembled those of rickets, the diagnosis of rickets was made in spite of the fact that the victims were dying of Teschen's disease. Trefny (1930) described a mass epidemic of some disease the course of which resembled that of Teschen's disease. He attributed it to some kind of vitamin deficiency and potato poisoning.

All these mistaken opinions were due to the fact that during the rounds made by the veterinarians, sick animals in the advanced paralytic stage had normal temperatures, and autopsy failed to disclose patholgic changes justifying the grave clinical picture. The prodromal stage usually characterized by elevation of temperature had somehow escaped the veterinarian's attention.

I presume that the owners called the veterinarian only at the late stage of the disease when motor nerve disturbances were the prevailing symptom, and when the temperature (previously elevated) had returned to normal. Many veterinarians did not consider the disease contagious. Some others diagnosed it as [illegible]. It was only in 1930 that a widely spread epidemic of Teschen's disease attracted the attention of veterinarians and breeders, and the appropriate authorities became interested in the etiology of this disease. The research work was entrusted to me because I was known to have been previously engaged in scientific investigation of the clinical, anatomo-pathological, and particularly histological aspects of the central nervous system.

From the very beginning I found lymphocytic infiltrations chiefly in the gray matter of the spinal cord, base of the brain, and cerebellum; I found them as well in the membranes of the brain, the cerebellum, and the spinal cord.

The lymphocytic areas were found around blood vessels, and in the spinal cord they were disseminated mainly in the inner corners. There were degenerative changes in the ganglion cells.

The macroscopic anatomical pathological changes were not typical.

On the skin, particularly in those cases when the course of disease is prolonged, abrasions can be frequently found in the regions of the hoofs, head, joints, and other parts of the body. These abrasions are due to continuous friction of the skin against the floor as a consequence of muscle cramps of head, body and limbs.

Findings in the gastro-intestinal tract show a moderate degree of gastro-intestinal catarrh; the mucous membrane of the small intestines may show yellow spots and is usually puffy.

After experimentation with transmission of the disease from sick to healthy animals by infecting them with the cerebro-spinal substance, and after the establishment of the virus nature of the causative factor (Berkefeld's and normal N filters), a very important question arose: what is the mutual relation between Teschen's disease and swine plague?

In 1933, I suggested to the Veterinary Department of the Ministry of Agriculture that Teschen's disease be officially

acknowledged as a separate entity and put on the list of diseases that must be compulsorily reported. This would enable us to combat this disease by applying to it the regulations enforced for the control of plague -- with some modifications as the two diseases are caused by different viruses. Further investigations have substantiated my opinion that Teschens disease is not identical to swine plague. Animals which recovered from Teschen's disease or were immunized against it, easily and often fatally succumb to the plague virus. I first came to this conclusion in 1933. This observation was the subject of studies made by Koscianski in Czechoslovakia in 1947, and in Germany by Fortner in 1942. Both investigators came to the conclusion that hogs who recovered from Teschens disease or were immunized against it displayed no resistance to artificial inoculation of the plague virus.

Here, I believe, it is important to remember that the danger of an infection by contact is much smaller in Teschens disease than in swine plague. For this reason, Teschens disease is much easier to bring under control than is swine plague by enforcing sanitary regulations. The statement made on page 112 of Veterinary Medicine, (1949, column 2, lines 27-28), to the effect that Teschen's disease differs from swine plague inasmuch as it is not transmitted by contact between sick and healthy animals, is incorrect. Teschen's disease, aside from other means of spreading, can be transmitted from the sick to the healthy animals through direct contact. In Macek's opinion, the next important problem is the relation of Teschen's disease to hog grippe. In 1937 Macek laid down his thesis that the term hog grippe should include enzootic bronchopneumonia, streptomycosis, colibacillosis, Teschen's disease, fibrous inflammation of other serous membrances

and joints, paratyphoid, and the disease which frequently has been diagnosed as hog murrain.

With reference to the Aujeszky's disease, Macek writes: "To my knowledge, we find descriptions of the disease only in our professional literature, but a positive diagnosis has never been made. According to these descriptions, the clinical picture of this disease resembles the description of some grippe epidemics." Macek claims that there were successful attempts to produce a typical Aujeszky's syndrome in experimental animals by means of the grippe virus. Macek says further that the grippe is a virus infection caused by a polyorganotropic virus but not a neurotropic, nor a pneumotropic one.

According to Macek's definition, a virus can be called neurotropic if from the beginning of the disease the central nervous system has been the only site of the pathological process, and there are no changes in other organs. All virus diseases belonging in the group of neurotropic viruses present a picture of inflammation without suppuration in various parts of the nervous system. This is also characteristic of exanthematous virus diseases.

Macek thinks that evidence of a neurotropic virus in this case can be found in the fact that the observed changes are irreversible and the destroyed cells never regenerate. In case of hog grippe, says Macek, we cannot speak of a pathological process attacking primarily or exclusively the central nervous system because other organs (lungs, intestinal tract, lymph nodes, skin) are affected simultaneously, or the disease has a pyemic character.

The above reasoning seems to suggest that Macek has a tendency to include Teschen's disease in the "hog grippe" group. However, on the basis of experimental observations accumulated so far, one cannot possibly share this opinion. Macek has not provided experimental support to his thesis so far. Many other investigators (Hruska, Zofijewski, Kosciansky) who had worked on this problem did not agree with Macek's opinion regarding hog grippe.

Further experiments will, no doubt, help to clear the confusion surrounding the problem of virus diseases in hogs.

THE INTERFERENCE PHENOMENON IN TESCHEN'S DISEASE

In GSR attention has been directed toward clearing of certain epizootiological phenomena accompanying the appearance of Teschens disease. Special attention has been devoted to the phenomenon of interference and to the concepts of Crawford; Harnach devoted himself to the task of investigating this phenomenon with regard to immunization procedures against erysipelas.

In a number of experiments on immunization against swine erysipelas, Pokorny, Tlusty, Zdziardzil, and Galliou found a decrease in the number of cases of Teschens disease during the vaccination period from approximately 3.8 percent to 1.35 percent -- in other words, a decrease of 2.45 percent (Harnach). In Harnach's experiments, out of a total of 10 hogs which were inoculated against erysipelas by a gonacrine type vaccine and then infected intranasally with a 20 percent cerebrospinal emulsion, only two developed Teschen's disease, whereas eight remained healthy.

Three hogs of the control group developed Teschen's disease. Harnach explains these results by the presence of a cellular resistance in the sense of the phenomenon of interferences. The intranasal inoculation procedure in the described cases took place within 20 to 60 days after vaccination against erysipelas; in other words, a long time after vaccination. In one case of a suckling pig infected with a virulent cerebrospinal emulsion of Teschens virus 14 hours after inoculation against erysipelas, a fully developed clinical picture of Teschens disease was observed. This means that during the 14 hours that had passed from the application of the gonacrine vaccine no resistance against Teschens disease had developed.

TREATMENT OF TESCHEN'S DISEASE

Different therapeutic measures have been used against this disease, particularly during the period around 1930 when the disease was first registered. But even then it was observed that recovery occurred in treated and untreated cases as well.

The methods of treatment used by veterinarians during the period preceding the year 1933, when the etiology was not yet known, varied. Metabolic drugs were used such as Fowler's arsenical solution, Carnofer, Pilisan, cod liver oil, Ephedrin -- Merck; Calcium preparations -- alone or in combination with adrenaline; sedatives such as chloral hydrate, opium, and bromides; laxatives; disinfectants, ~~anthelmintics~~ (chenopodium, kebal, calomel, semen arice, isticin, castor oil), Lugol solution, Methyleneblue; cardiac drugs and diuretics (caffeine, digipurate, etc.).

With regard to bacteriological findings, sera were used (antistaphylococcic, antihemorrhagic). For non-specific protein therapy, horse serum or measles serum were used.

Other than pharmacological means of treatment used: diet, change of food, hygienic measures of precaution, rest, dark room, warmth in winter.

These treatment procedures were administered without any system, without knowledge of the course of disease, of pathological anatomy of the nervous system, and of epidemiology. It soon became evident that a great number of sick animals did not respond to these methods of treatment. If we consider the nature of the pathological process in the central nervous system, we can easily understand the therapeutic difficulties in the case of an advanced stage of the disease. It came to a point when all therapeutic methods were given up, and the affected hogs were being sent to slaughterhouses from necessity. Owners were granted indemnity. As early as 1944 I noticed that animals after spontaneous recovery developed an immunity against reinfection and displayed a tolerance of a virulent virus emulsion administered intracerebrally. This observation gave hope for an effective preventive action by administration of preventive inoculation against Teschenk disease. To the solution of this problem I have devoted a great deal of work. I dwelt previously on this subject in this paper.

The fact that for some time the official veterinary authorities ordered compulsory slaughter of all affected animals made it difficult to obtain exact information about the natural course of the disease in most cases. The prevailing opinion was that in our fight

against this disease we are helpless, and have no means to exert influence upon the course of it. This opinion turned out to be wrong as we can see now.

In view of the fact that we still have no effective method of treatment, the official regulations enforcing slaughter of affected animals must at present be looked upon as justified.

It might be of interest to give a brief review of research work I did in 1947 on the problem of obtaining a therapeutically effective serum against Teschen's disease.

This serum was derived from hogs hyperimmunized against Teschen's disease. It was prepared in 1940; it would be reasonable to assume that its effectiveness was considerably weakened by 1947. Altogether, I tried it out on 117 hogs.

In the control group of 32 affected but not treated animals spontaneous recovery amounted to 9.35 percent; slaughtered 15.62 percent; fatality rate was 75 percent; total losses were 90.62 percent.

In the group of 68 hogs affected with the disease and treated with serum: recovery 27.04 percent; slaughtered 29.42 percent; died 42.61 percent; total losses were 72.06 percent.

In the group of 17 healthy animals brought from farms recently stricken with the disease and prophylactically inoculated with serum; 29.41 percent of these took sick and 70.59 percent remained healthy. The total losses in this group were 11.76 percent; the number of those that did not develop the disease plus the number of recoveries was calculated as 88.24 percent.

In the group of 6 young hogs brought from farms affected by the epidemic, but still without signs of disease and not inoculated prophylactically, there was only one case of disease, which is equivalent to 16.66 percent; 83.34 percent remained healthy.

The sick hog recovered, so that in this small group without prophylactic inoculation and without treatment there was a positive result of 100 percent. This small group cannot be considered qualified for comparison with the former groups because of the small number of animals included in observation. The good health of the animals in this particular group might also have been a result of better hygienic conditions.

Good nourishment was an additional therapeutic agent. During the paralytic stage of disease sick animals could not receive their nourishment without help. Consideration must be given to this condition. In such cases the best feed is milk or cereal with milk, bran, boiled potatoes, as swallowing might be impaired in some cases.

Care should be taken, particularly in winter, to keep the animals warm and dry.

In some cases the recovery of the animal is not complete. Complications in the form of contractures of muscles or limbs, or paralysis of the back, etc., may occur. Prolonged care given to such animals involves danger of keeping the source of infection open which might contribute to further spread of the disease. The amount of work with the resultant waste of time and expense involved in treating these sick animals is considerable.

We cannot claim positive results of treatment in cases like these.

For this reason, it is recommended that animals which are still fit for the meat market be sent to slaughterhouses. Slaughter on the premises should not be done so that further dissemination of infection will be prevented.

Good signs which give reason to expect recovery of the animal are: improvement of appetite and tendency to maintain normal body temperature.

Unfavorable symptoms are: drop of body temperature below normal and lack of appetite.

The above-mentioned experimental data seem to encourage the use of sera for therapeutic purposes. Unfortunately, the therapeutic effect is not so reliable as to warrant the recommendation of this method for wide use by practitioners. Therefore, slaying or slaughter of sick animals is under the circumstances the only method recommended to combat the spread of the epidemic.